

# CASE REPORTS

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## Psittacosis

### *Clinical Presentation and Therapeutic Observations*

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PSITTACOSIS is an uncommon cause of acute respiratory disease acquired after inhalation of dry excreta from infected psittacine birds, primarily parakeets and parrots. The disease in humans ranges from a mild febrile illness to a systemic infection resembling a severe influenza-like syndrome. Recently, there has been a progressive increase in reported cases per year with most patients giving a history of direct contact with treated and, presumably, noninfected caged birds.<sup>1</sup> We report a typical case of psittacosis, describing the usual clinical manifestations and commenting on the possible reasons for an increasing incidence.

### Report of a Case

A 57-year-old previously healthy woman was admitted to hospital after a five-day history of shaking chills and high spiking temperatures, mild nonproductive cough, anorexia, nausea and vomiting. She said she had not recently traveled out of the country or been exposed to any ill family members.

On the initial physical examination the patient was very lethargic and confused, with a temperature of 40°C (104°F), a heart rate of 120 beats

per minute, blood pressure of 120/70 mm of mercury supine, decreasing to 80/50 sitting. Examination of the chest disclosed decreased breath sounds, dullness to percussion and rales over the right posterior hemithorax. Other examination findings were within normal limits. Routine laboratory data disclosed the following values: Hemoglobin, 16 grams per ml; hematocrit, 48 percent, and leukocyte count, 9,800 per cu mm. The platelet count was within normal limits. The serum sodium was 148 mEq per liter; potassium 3.1 mEq per liter; chloride 117 mEq per liter; bicarbonate 22 mEq per liter; blood urea nitrogen (BUN) 15 mg per dl; blood glucose, 203 mg per dl, and alkaline phosphatase, 51 IU per liter. Serum glutamic oxaloacetic transaminase (SGOT) was 48 units per liter; lactic dehydrogenase (LDH), 293 units per liter, and total bilirubin, 0.4 mg per dl. Arterial blood gases drawn while the patient breathed room air showed a pH of 7.50, oxygen pressure (Po<sub>2</sub>) of 83 and carbon dioxide pressure (PCO<sub>2</sub>) of 19 mm of mercury. A roentgenogram of the chest showed a consolidating right lower lobe infiltrate with a small pleural effusion (Figures 1 and 2). Thoracocentesis disclosed 1,600 leukocytes per cu mm with a differential count of 17 percent polymorphonuclear leukocytes, 54 percent lymphocytes and 29 percent mesothelial cells. Pleural fluid LDH was 198 units per liter and pleural fluid protein was 210 mg per dl. No organisms were seen on Gram stain or acid-fast bacillus stains. Induced sputums resulted in non-specific findings.

The patient was given 500 mg of erythromycin intravenously every six hours because Legionnaires' disease was suspected. She slowly improved while remaining febrile through the fourth hospital day. At this time repeat serum chemistries showed alkaline phosphatase, 162 IU per liter; SGOT 260 units per liter; LDH, 705 units per liter, and total bilirubin, 0.8 mg per dl.

A thorough history taken again at this time disclosed that the patient had recently purchased a parrot that died after a brief respiratory illness, approximately ten days before the onset of her symptoms.

The patient was discharged in good condition after a ten-day stay in hospital. She continued

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to take erythromycin orally for one more week. A month after presentation the patient felt well, all laboratory values had returned to normal and a roentgenogram showed that the consolidating pneumonia had resolved. Acute and convalescent serum titers for Legionnaires' disease were negative; however, complement-fixation titers for chlamydiae had a significant fourfold increase from less than 1:5 to 1:128 within two weeks. Close family members not residing with the patient had negative titers for chlamydiae.

The patient's first parrot and a second one subsequently purchased from the same pet store by her family died of a respiratory illness. Both birds showed classic findings of systemic psittacosis at autopsy and *Chlamydia psittaci* was isolated in cultures grown from specimens from each parrot.

### Discussion

Psittacosis in humans results from inhaling *Chlamydia psittaci*, Gram-negative obligate intracellular organisms capable of independent metabolism. They have a discrete cell wall and are susceptible to antibiotics.<sup>2</sup> The common reservoirs for these organisms are psittacine birds (parrots, parakeets and cockatoos), with the more inclusive term *ornithosis* indicating that non-psittacine species (pigeons, turkeys and ducks) are also potential carriers, especially in the United States. Most published cases have resulted from epidemics,<sup>3,4</sup> although in this country most cases are of a sporadic nature.<sup>1,5</sup> The estimated mortality ranges from 1 percent to 5 percent, with parrot and turkey strains being more virulent for humans. However, appropriate therapy in patients in whom the disease has been identified early can result in a fatality rate of less than 1 percent.<sup>6</sup> Most patients relate a history of bird exposure,<sup>1,5</sup> although the responsible animals may not have appeared sick or may have had only a mild illness. Person-to-person transmission has been reported, but is apparently rare.<sup>7</sup>

The disease typically presents, after an incubation of one to two weeks, as an acute illness manifested by a nonproductive cough, dyspnea, high spiking fevers, often greater than 40.6°C (105.1°F), and severe diffuse headaches. Frequently, complaints of pleuritic chest pain, blood-tinged sputum, myalgias and arthralgias are noted.<sup>3,5</sup> Patients with severe cases invariably have pneumonia, and some have clinical symptoms resembling typhoid fever, including the presence of bradycardia and rose spots.<sup>5,6</sup> Other

significant complications may include invasion of the central nervous system with delirium and stupor being noted, as well as pronounced hepatic involvement during the initial septicemia which can result in features similar to those of acute

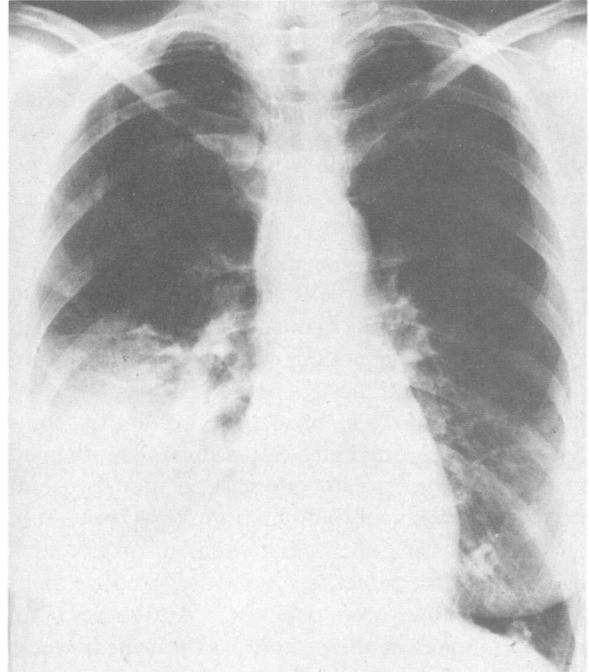


Figure 1.—Posteroanterior roentgenogram of the chest showing consolidating right lower lobe infiltrate.

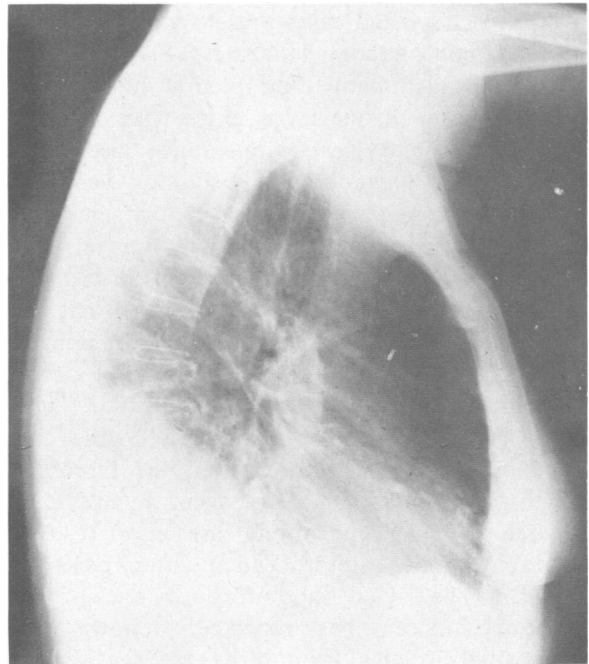


Figure 2.—Lateral roentgenogram of the chest showing right lower lobe infiltrate.

viral hepatitis. Occasionally, pericarditis and fatal myocarditis have been reported.<sup>3,5</sup> Physical signs of pulmonary consolidation are frequent, with pleural friction rubs being reported in a number of patients. Hepatosplenomegaly is often described and occurs in up to 30 percent of the patients. Pericardial friction rubs, cervical adenopathy, pharyngitis and erythema nodosum have also been noted.<sup>3,5,7</sup>

Laboratory data typically are nondiagnostic, with leukocyte counts often being normal or slightly increased with a nonspecific differential. Liver function abnormalities resembling viral hepatitis have also been reported.<sup>5</sup> Sputum, if productive, is also nonspecific with only a few polymorphonuclear cells being seen on Gram stain, although the organisms may be isolated in two to 30 days depending on the sophistication of the culture techniques.<sup>2</sup> The complement-fixation test is group-specific, measuring antibodies common to all chlamydiae. Seroconversion, defined as a fourfold or greater increase in titer, is useful in the diagnosis of lymphogranuloma venereum and psittacosis. Other chlamydial disorders such as genital-tract infections typically produce non-diagnostic low titer changes.<sup>2</sup> Antibodies that usually appear in the second to the fourth week, however, may be delayed or suppressed if antibiotic therapy is initiated.<sup>7,8</sup>

Roentgenographic findings are variable. However, most reported cases present with segmental or lobar consolidating infiltrates (Figures 1 and 2). Hilar adenopathy and pleural involvement were common in one large series with a homogeneous "ground glass" appearance also being reported.<sup>9</sup> Roentgenographic resolution was often delayed averaging over six weeks before significant clearing occurred.<sup>3,9</sup> Drug therapy for this disorder has not been thoroughly evaluated, although tetracycline in a dosage of 2 grams per day for two weeks is effective with chloramphenicol being the alternate drug of choice.<sup>7</sup> Indolent responses to tetracycline are often noted,<sup>5,8</sup> possibly related to the various dosage schedules that have been advocated throughout the literature. Erythromycin is an effective agent against chlamydiae in vitro,<sup>10</sup> and in one case report resulted in rapid clinical improvement after penicillin failed.<sup>11</sup> The avian laboratory of one of the authors (R.E.D.) has repeatedly shown that chlamydial isolates from birds are sensitive to erythromycin and are eradicated effectively after a 21-day course of therapy.<sup>12</sup> Our patient, despite

the lack of serological evidence of Legionnaires' disease, also showed a rapid response to erythromycin. Of note are recent reports emphasizing the close antigenic relationship between Legionnaires' disease and chlamydial psittacosis<sup>13</sup> and their similar clinical presentation.<sup>10</sup> Because a clinical distinction between these two unusual respiratory infections is impossible at present, a thorough history of bird exposure is essential to an early diagnosis. Serological testing would help determine the prevalence of these disorders in various communities. Further studies are needed to determine the usefulness of erythromycin in controlling suspected cases of either disorder.

A more important issue than which antibiotic to use for this disease is how to successfully identify, isolate and treat all infected birds entering this country. Present policies have resulted in the widespread release of birds infected with chlamydiae from quarantine stations despite attempts to administer antibiotics. The drugs are often given in a diet meal foreign to most birds,<sup>12,14</sup> resulting in the ingestion of subtherapeutic doses of chlorotetracycline.<sup>12</sup> Continuation of ineffective therapy of infected birds while in quarantine is likely to result in increased dissemination of psittacine disease when the birds are released. This is substantiated by recent increases in reported human cases.<sup>1</sup> Early recognition of this disorder is crucial for the control of epidemics, the rendering of appropriate therapy for sporadic cases, and the identification and education of quarantine station and pet store personnel who may be releasing infected birds.

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